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**Amendments to the Specification:**

Please replace the paragraph at page 232, lines 3-20 with the following amended paragraph:

The GAGE-1 gene was identified previously as a gene that codes for an antigenic peptide, YRPRPRRY (SEQ ID NO:1392), which was presented on a human melanoma by HLA-Cw6 molecules and recognized by a clone of CTLs derived from the patient bearing the tumor. By screening a cDNA library from this melanoma, De Backer O, et al. (*Cancer Res* 1999 Jul 1;59(13):3157-65) identified five additional, closely related genes named GAGE-2-6. We report here that further screening of this library led to the identification of two more genes, GAGE-7B and -8. GAGE-1, -2, and -8 code for peptide YRPRPRRY. Using another antitumor CTL clone isolated from the same melanoma patient, they identified antigenic peptide, YYWPRPRRY (SEQ ID NO:1393), which is encoded by GAGE-3, -4, -5, -6, and -7B and which is presented by HLA-A29 molecules. Genomic cloning of GAGE-7B showed that it is composed of five exons. Sequence alignment showed that an additional exon, which is present only in the mRNA of GAGE-1, has been disrupted in gene GAGE-7B by the insertion of a long interspersed repeated element retroposon. These GAGE genes are located in the p11.2-p11.4 region of chromosome X. They are not expressed in normal tissues, except in testis, but a large proportion of tumors of various histological origins express at least one of these genes. Treatment of normal and tumor cultured cells with a demethylating agent, azadeoxycytidine, resulted in the transcriptional activation of GAGE genes, suggesting that their expression in tumors results from a demethylation process.

Please replace the paragraph at page 671, line 10 through page 672, line 4 with the following amended paragraph:

The S6/H4 kinase purified from human placenta catalyzes phosphorylation of the S6 ribosomal protein, histone H4, and myelin basic protein. In vitro activation of the p60 S6/H4 kinase requires removal of an autoinhibitory domain by mild trypsin digestion and autophosphorylation of the catalytic domain (p40 S6/H4 kinase). The two autophosphorylation/autoactivation sites contain the sequences SSMVGTPY (site 1) (SEQ ID NO:1394) and SVIDPVPAPVGDSHVDGAAK (site 2) (SEQ ID NO:1381). These sequences



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identify S6H4 kinase as the rac-activated PAK65 (Martin, G. A., Bollag, G., McCormick, F. and Abo, A. (1995) EMBO J. 14, 1971-1978). Site 1 phosphorylation is most rapid, but activation does not occur until site 2 is autophosphorylated. The site 1 phosphorylation occurs by an intramolecular mechanism whereas site 2 autophosphorylation occurs by an intermolecular mechanism. A model is proposed in which phosphorylation of sites 1 and 2 occurs sequentially. The model proposes that trypsin treatment of the inactive holoenzyme removes an inhibitory rac-binding domain which blocks MgATP access to the catalytic site. The pseudosubstrate domain at site 1 is autophosphorylated and subsequent bimolecular autophosphorylation at site 2 fully opens the catalytic site. Phosphorylation by a regulatory protein kinase may occur at site 2 in vivo.

Please replace the paragraph at page 724, lines 6-20 with the following amended paragraph:

The S6/H4 kinase purified from human placenta catalyzes phosphorylation of the S6 ribosomal protein, histone H4, and myelin basic protein. In vitro activation of the p60 S6/H4 kinase requires removal of an autoinhibitory domain by mild trypsin digestion and autophosphorylation of the catalytic domain (p40 S6/H4 kinase). The two autophosphorylation/autoactivation sites contain the sequences SSMVGTPY (site 1) <u>(SEQ ID NO:1394)</u> and SVIDPVPAPVGDSHVDGAAK (site 2). These sequences identify S6H4 kinase as the rac-activated PAK65 (Martin, G. A., Bollag, G., McCormick, F. and Abo, A. (1995) EMBO J. 14, 1971-1978). Site 1 phosphorylation is most rapid, but activation does not occur until site 2 is autophosphorylated. The site 1 phosphorylation occurs by an intramolecular mechanism whereas site 2 autophosphorylation occurs by an intermolecular mechanism. A model is proposed in which phosphorylation of sites 1 and 2 occurs sequentially. The model proposes that trypsin treatment of the inactive holoenzyme removes an inhibitory rac-binding domain which blocks MgATP access to the catalytic site. The pseudosubstrate domain at site 1 is autophosphorylated and subsequent bimolecular autophosphorylation at site 2 fully opens the catalytic site. Phosphorylation by a regulatory protein kinase may occur at site 2 in vivo.